# INDUSTRIAL HYGIENE SECTION

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Industrial Medicine

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# The Toxicity of Antimony

## —*Animal Studies*—


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LITTLE is known concerning the toxicity of metallic antimony and its compounds when used industrially.

Perhaps the first industrial employment of the metal dates back to the Chaldeans, who used it about 4000 B.C. in the manufacture of vessels and vases. Archeological discoveries show that other ancient civilizations made use of both the metal and the black sulfide. The history of antimony has been excellently reported in the comprehensive brochure by Habeck[1] (1936). Examination of the extensive literature on these materials and other antimony compounds clearly indicates their widespread industrial utilization at the present time.

In the United States, industrial workers are exposed to antimony in the mining of the ore, the smelting, refining and utilization of the metal, and the production and use of its compounds. The metal is employed especially as an alloy with other metals and in particular with lead, tin and copper. Nearly all industrial lead alloys contain antimony. Chief among these are bearing metals, type metals, storage battery grids and pewters. Antimony is a strategic metal for national defense, being incorporated in the manufacture of munitions, tools and machines. The development of domestic antimony sources, enlarging ore reserves and increasing domestic smelter capacities as a national defense measure has progressed rapidly in the last few years.

Industrial processes present the opportunity for worker absorption of antimony in the form of


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fumes and dust through the respiratory passages and in the digestive tract. The literature reports[2] that absorption may take place also through the skin. It is held by some authorities that the chief danger to workers with antimony alloys is from lead, arsenic or other metals which they contain. However, many cases of occupational illness attributable directly to the manufacture and use of antimony and its compounds as well as its alloys are reported in the literature together with specific clinical symptoms of poisoning in both the acute and chronic forms.

This paper presents, first, a brief historical survey of the pertinent literature, and second, laboratory studies on the toxicity of antimony and five of its compounds. The experimental work represents utilization of 357 small animals in the antimony study and 160 animals in a comparative lead study. Included in the comparative work is a study of the minimum fatal intraperitoneal dose for lead metal and five of its compounds. In the antimony phase of the work the minimum fatal intraperitoneal dose was determined for rats, together with the resultant pathology in the fatal episode and in sub-lethal dose survivors. Rats and rabbits were fed antimony metal and tartar emetic for periods up to 12 months. Minimum fatal dose determinations were made of the metal and antimony potassium tartrate by mouth in rats and intraperitoneally and by mouth in guinea pigs. Hematological, gross and microscopic pathological examinations, chemical and spectrographic analyses were made on representative animals. The subjection of small animals to the inhalation of antimony dusts and fumes has, up to now, not been undertaken.

**Historical**

CAROZZI[2] in 1930, White[3] in 1934 and McNally[4] in 1937 have presented literature summaries of many industrial antimony poisoning cases together with clinical findings dating from the first description of the effects of antimony on chemists by Ramazzini[5] about 1700. Included in these reports are the findings of Schrumpf and Zabel,[6] 1910. They noticed among younger workers in a type foundry very few cases of lead poisoning, but a number with: "remarkable facial expression, complaints of nervousness, irritability, sleeplessness, fatigue, dizziness, headache, both frontal and cerebral, muscular pain, neuralgia in the extremitiesties, nausea, loss of appetite, gastro-intestinal disturbances and constipation." Examination of the blood revealed a diminished leucocyte count and a notable eosinophilia. A lowering of the blood pressure was noted. Antimony was found in the fecal discharges. The symptoms disappeared after suspension of work for two to three weeks. Experimental feeding of rabbits with antimony oxide and sulfide produced similar changes in the blood.

Selisky[7] in 1928 reported 200 cases of industrial skin lesions due to the use of a solution of antimony salts as a mordant in cloth dyeing. He described a pustular necrotic dermatitis commencing as a folliculitis and ending with atrophic scarring and cites intermediate products of the antimony salts as the etiologic factor. He further noted the condition to be worse in the summer months. Oliver[8] (1933) found among antimony trioxide workers occasional skin irritation, a slight lowering of the blood pressure and a marked daily excretion of antimony in the feces, but concludes that the occupation has little danger.

However, Feil[9] in 1939 reported considerable illness among workers in an antimony foundry. He states, "among 15 workers exposed to vapors (the white oxide and metal vapors produced in the melting process) seven men had a rash when examined and 14 had had it at some time. It consisted of pustules very similar to smallpox. The neck, forearms and legs were usually affected. In some persons the rash covered almost the whole body, while in others there were few pustules. They arose after a few weeks' work and healed when the worker left the job. The only worker who had never had the rash suffered greatly, however, from the vapors (lack of appetite, headache, oppression). Half of the workers had conjunctivitis and half had anemia and other complaints."

Factory inspection reports from Hamburg, Germany, showed that the workers grinding metallic antimony frequently suffered from dyspepsia, headache, vomiting, conjunctivitis and a bloody discharge from the nose. Dutch inspectors in Holland found what they held to be acute antimony poisoning in a man who broke up large pieces of the metal. It nevertheless appears that the opinion of authorities[2] is divided on the possibility that antimony may cause industrial poisoning.

A few toxicological studies dealing with industrially used antimony compounds have been reported. In 1912, Boveri[10] fed rabbits metallic antimony suspended in oil in doses from five to 55 milligrams every other day for 30, 60, and 90 days without apparent bad effects. Larger doses produced diarrhea with a progressive cachexia and death. Abortions were frequent. Autopsies revealed hypertrophy of the heart and adrenals. The aorta and other arteries showed no alterations such as are found in lead poisoning.

Seitz[11,12] in 1923 and 1924 corroborated his blood findings among type founders with those he obtained experimentally through subcutaneous injections of antimony in cats. A diminution in red and white blood cells resulted. Antimony administered by stomach caused a diminution of white cells and an increase in red cells. The results of experiments on guinea pigs were not so pronounced.

Manson[13] in 1926 conducted electro-cardiographic experiments following intravenous injections of antimony at doses of one-tenth to one-half minimum fatal doses. A slowing of rate, lengthening of atrio-ventricular conduction and arrythmia resulted and he concluded that the fundamental function of the heart muscle was affected and the action was local in the myocardial tissue and not in the inhibitory centers. A marked lowering of blood pressure also occurred.

Maneghetti[14] in 1926 injected trivalent antimony

(colloidal antimony sulphide and tartar emetic) slowly into the veins of rabbits. This caused a "tone blood disease" characterized by the appearance in the circulation, within 24 hours of numerous normoblasts, polychromatic erythrocytes, Howell-Jolly bodies, red cells with basophilic stippling and an increase in reticulated erythrocytes. There was leucocytosis with the presence of myelocytes. The red blood cell count and hemoglobin remained normal. After 200 hours, there was a lowered red blood cell count, a drop in hemoglobin and intense anisocytosis with many large normocytes. Normoblasts were also present. The rapidity of injection had a marked effect on the intensity of the blood changes, the degree of toxicity and the localization and nature of pathological changes.

Flury[15] (1927) conducted feeding experiments on rats, cats and dogs using tartar emetic, antimony trioxide and antimony pentoxide. He found that doses of the trioxide and pentoxide as high as four milligrams daily could be fed to 150 gram rats without appreciable effect on growth. Daily doses of 200 milligrams of the trioxide resulted in slight irregularities in the growth of rats while death resulted with the same dose of tartar emetic. Two cats sickened and lost weight after the continued feeding of these oxides at 450 milligram doses daily. The pentoxide was found to be slightly less toxic than the trioxide. Dogs were more sensitive than small rodents and showed digestive upsets, vomiting and diarrhea after the administration of only a few grams of material. Tartar emetic was found to be by far the most toxic of the compounds studied. About 10 milligrams per kilogram caused vomiting in cats and four milligrams per kilogram produced similar effects in dogs. Continued feeding of small amounts of antimony compounds including tartar emetic hindered the growth of young rats; with larger doses definite retardation of growth occurred. No certain evidence of tolerance to antimony was obtained.

In 1926, Pribyl[16] found an increase in the nonprotein nitrogen of the blood in the experimental subacute tartar emetic poisoning of four rabbits. This he believed is due to a rise in urea nitrogen and is associated with an increase in this substance in the urine. The ammonia nitrogen quotient in the urine of poisoned rabbits seems to be inversely proportional to the urea nitrogen quotient. The acceleration of protein metabolism is attributed mainly to increased autolysis of tissues.

Lucia and Brown [17] in 1934 injected a freshly prepared 1% solution of tartar emetic intravenously in rabbits. A six-milligram per kilogram dose reduced the leucocyte count by 50% within five minutes. No subsequent leucocyte loss was observed in 28 hours. Normoblasts were moderately increased while the blood platelet count remained unchanged.

Habeck[1] in 1936 reported a marked lowering of the leucocyte count in chronic antimony poisoning.

Oelkers[18] who in 1937 discussed the pharmacology of antimony, states that it is a general cell poison whose action disturbs "vascular breathing," checks albumen putrefaction and fat and carbohydrate metabolism. He further states that the view held by many that antimony causes capillary paralysis is not true. He found[19] that when rabbits were poisoned with tartar emetic, a considerable disturbance in the catabolism of glucose and alcohol resulted.

In 1923 Brahmachari[20] observed that guinea pigs could not survive intramuscular injection of a glycerine solution of antimony trioxide in doses of 25 milligrams per kilogram but survived 20 milligram doses.

Carlson [21] found that 8% of the antimony in the crimson sulphide and about 3% of the antimony in the golden sulphide was soluble in gastric juice. He considered it possible that this solubility was sufficient to be a source of danger to men exposed to these dusts.

Bachem[22] has shown that chronic ingestion of antimony produces "serious degeneration of the internal organs" without establishing a tolerance.

## Experimental

MINIMUM LETHAL INTRAPERITONEAL DOSE IN RATS: One hundred and forty nine albino rats were used to determine the minimum lethal intraperitoneal dose for antimony metal and five of its compounds frequently used in industrial practices. The relative toxicities of these materials were sought by implantation of known quantities in the peritoneal cavities of the animals. The materials were finely ground and passed through a 325-mesh sieve to effect a small particle size and thus aid their absorption within the body. The slightly soluble compounds were incorporated in fresh corn oil, the soluble compounds in distilled

TABLE I.

MINIMUM LETHAL DOSE FOR ANTIMONY COMPOUNDS ADMINISTERED INTRAPERITONEALLY TO RATS

| Material | | M.L.D./50 Milligrams of Antimony per 100 Grams Body Weight | Total Weight of Compound in Milligrams |
|---|---|---|---|
| Antimony potassium tartrate | $KSbOC_4H_4O_6 \cdot \frac{1}{2}H_2O$ | 1.1 | 3 |
| Antimony metal | Sb | 10.0 | 10 |
| Antimony trisulfide | $Sb_2S_3$ | 100.0 | 139 |
| Antimony pentasulfide | $Sb_2S_5$ | 150.0 | 247 |
| Antimony trioxide | $Sb_2O_3$ | 325.0 | 389 |
| Antimony pentoxide | $Sb_2O_5$ | 400.0 | 531 |
| Controls, corn oil | | Survived | 3 ml. |
| Controls, distilled water | | Survived | 3 ml. |
| Controls, untreated | | Survived | |

water. Many metallic materials, when suspended in corn oil, catalyze its oxidation and produce organic acids which convert insoluble compounds to soluble ones. This effect increases the toxicity of the compound and leads to erroneous conclusions. Accordingly, only fresh suspensions of substances in fresh corn oil were used. All dosage was based upon the percentage of antimony in the compound in order to facilitate comparison with the toxicity of other metals and metal compounds. Three groups of control animals were established. One group received corn oil intraperitoneally, a second group distilled water, while a third group remained untreated.

In Table I the antimony compounds used are listed in the order of their toxicity. The minimum lethal dose presented in column two is the dose which killed 50% of the animals. Column three is a calculation from column two showing the corresponding entire weights of the compounds injected.

The animals represented by this phase of the study were adult, healthy albino rats which met standards for normalcy previously established for this strain. All laboratory animals received high caloric, high vitamin diets augmented by lettuce twice weekly. The materials used were of high purity, caution being observed that any arsenic content was negligible. Postmortem examinations were made of all animals to determine the cause of death in the acute episode and to show the response of body tissue to absorption of sub-lethal antimony doses. The latter group of animals were sacrificed at various periods from 15 to 70 days following injection. Tissues from representative animals were examined miscroscopically.

Animals dying within a few days after injection showed dyspnea, loss of weight, general weakness, loss of hair and evidence of myocardial insufficiency. Predominant physical signs in survivors were marked immediate loss of weight with a retarded, gradual gain after five to 10 days and a marked loss of hair with a dry and scaly appearance of the skin. Hematological studies made of these animals showed an eosinophilia.

**Pathological Findings**

AT autopsy, gross findings in the acute episode were myocardial congestion with engorgement of cardiac blood vessels, dilatation of the right heart and little change in the lungs. The abdominal viscera were generally softened and congested with occasional interstitial hemorrhage. Numerous areas of the peritoneal surfaces were overlain with the injected material. Death was attributed to myocardial failure. In surviving animals the initial acute congestion had partially subsided when seen at autopsy, although cut sections of the hearts, livers and kidneys showed congestion and occasional signs of early degenerative changes. The small intestines usually were hemorrhagic. A fibrous tissue response in the abdominal cavity anchored peritoneal surfaces and walled off areas of the injected material in dependent portions.

Microscopic examination showed the lungs of all animals to be essentially negative. In those rats succumbing to lethal dosage, death was attributed to myocardial edema with marked hyperemia and capillary engorgement of the subendocardial zone. The livers showed moderate periportal congestion with vacuolization, degeneration and polymorphonuclear infiltration. Antimony oxides and sulfides produced no change in the spleen, but with the metal there was moderate hyperplasia with an increase in eosinophils. With the more soluble tartrate there was, in addition, an acute picture of intense splenic congestion. An acute toxic glomerulo nephritis (Fig. 1) with glomerular congestion, apparently albuminous material in the glomerular spaces and a degeneration of tubular epithelum also resulted in those animals receiving the tartrate. A similar response, but of mild degree, was found in rats receiving the lesser soluble materials.

In the microscopic examination of animals surviving sub-minimum lethal dosage more marked signs of chronic change were noticeable in the heart. A marked variation appeared in the staining of myocardial fibers. Intensely red staining cells, in which the fibrillar structure was indistinct, appeared more numerous in the sub-endocardial and epicardial zones. A fine stippling of dark staining pigment was present within numerous muscle fibers, more noticeable in animals receiving the metal (Fig. 2). Other myocardial cells were edematous. In the antimony potassium tartrate animals there was an increase in connective and fibrous tissues of the myocardium. In the livers (Fig. 3) of nearly all animals there was sub-capsular congestion and in some instances blood pigmentation. A mild hepato-toxemia was characterized by a functional hypertrophy of many cells displaying a cloudy appearance and finely

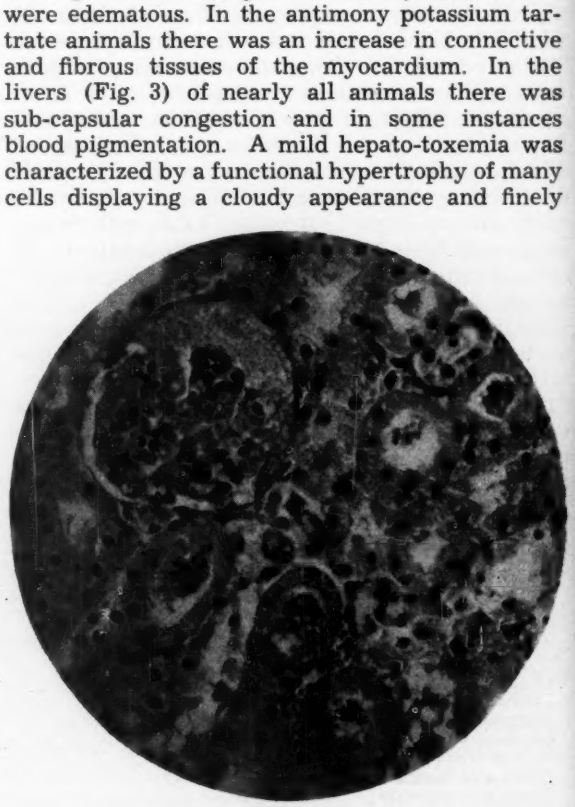

**FIG. 1.**
**High power photomicrograph showing kidney damage characteristic of animals receiving lethal doses of antimony metal or its compounds**

TABLE II.

MINIMUM LETHAL DOSE FOR ANTIMONY COMPOUNDS ADMINISTERED INTRAPERITONEALLY TO GUINEA PIGS

| Material | | M.L.D./50 Milligrams of Antimony per 100 grams Body Weight | Total Weight of Compounds in Milligrams |
|---|---|---|---|
| Antimony potassium tartrate | $KSbOC_4H_4O_6 \frac{1}{2} H_2O$ | 1.5 | 2.5 |
| Antimony metal | Sb | 15.0 | 15.0 |
| Control—calcium carbonate | $CaCO_3$ | Survived | 100 |
| Control—iron oxide | $Fe_2O_3$ | Survived | 100 |
| Control—corn oil | | Survived | 2 ml. |
| Control—distilled water | | Survived | 2 ml. |
| Control—untreated | | Survived | |

reticulated cytoplasm. Numerous plasma cells and deep staining cells with picnotic nuclei were in evidence. In addition there was a slight periportal connective tissue increase with capillary congestion and infiltration of a few lymphocytes in livers of animals injected with antimony potassium tartrate. The spleens were essentially negative with a slight congestion and a moderate diffuse hyperplasia in animals receiving the metal and tartar emetic. Glomerular congestion with coagulated, albumen-like material in the tubules was also seen in the kidneys of these latter animals.

**Comment**

THE above findings establish a relatively low order of toxicity for the oxides and sulfides of antimony. The minimum fatal dose for the antimony sulfides, injected intraperitoneally, appears to be one-third that for the respective oxides. Antimony metal, however, presents a rather high order of toxicity. The high toxicity of tartar emetic, 10 times that of the metal, long has been recognized. The toxemia evident in the livers and kidneys is not surprising since such a response is frequently associated with the elimination of toxic materials. The suggestion here is that an albuminurea may be present during antimony absorption. The rather specific attack upon the myocardium is conspicuous by its nature and consistency. In the medical examination of workers exposed to antimony and to metals incorporating antimony, attention to physical signs and symptoms related to the heart may be justified.

**Minimum Lethal Intraperitoneal Dose**

THE minimum lethal intraperitoneal dose for antimony metal and antimony potassium tartrate was determined on normal guinea pigs. Approximately the same ratio of toxicity, 1 to 10, exists between these materials as when administered to rats (Table II). Control pigs received calcium carbonate and iron oxide in doses of 100 milligrams per 100 grams of body weight with the expectable absorptive and inert responses respectively. Additional control animals as listed in Table II were without poisoning. Eighty-eight guinea pigs were used in this phase of the study. Gross pathological findings in these antimony-treated animals was in close accord with those found in similarly treated rats.

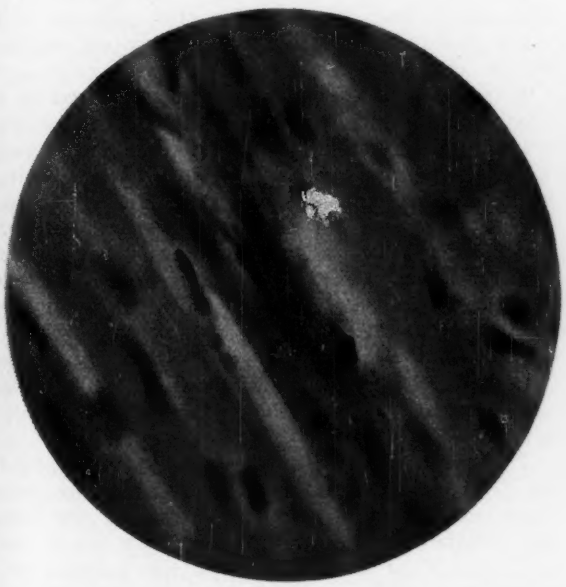

**FIG. 2.**
**Oil immersion photomicrograph showing fine pigmentation in myocardial tissue from a rat receiving a sub-lethal intraperitoneal dose of antimony metal**

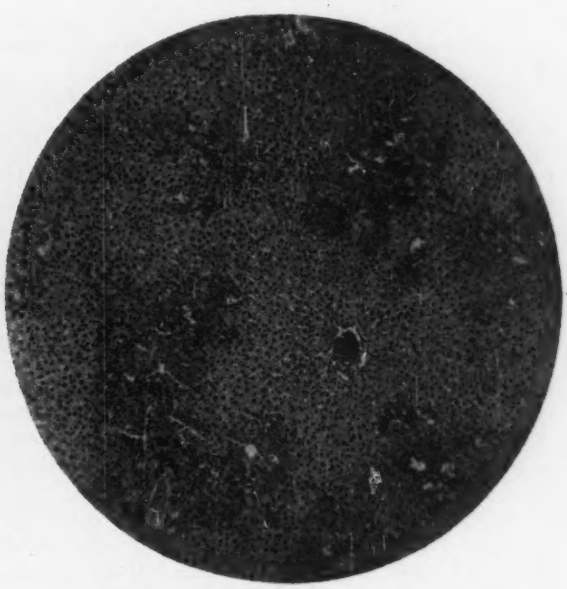

**FIG. 3.**
**Low power photomicrograph showing liver damage characteristic of rats receiving sub-lethal intraperitoneal doses of antimony potassium tartrate**

### Comparative Study of Similar Lead Compounds

IN view of the fact that lead and antimony are closely associated in industrially used alloys, it appeared wise to compare certain similar lead compounds with antimony compounds. In this comparative study the minimum lethal dose of these lead compounds was determined by intraperitoneal injection into albino rats. Similar procedures were employed as when determining the minimum lethal dose for antimony compounds. One hundred and fifty animals received doses calculated as milligrams of lead present in the compound per 100 grams of animal body weight (Table III).

TABLE III.

MINIMUM LETHAL DOSE OF LEAD COMPOUNDS ADMINISTERED INTRAPERITONEALLY TO RATS

| Material | | M.L.D./50 Milligrams of Lead per 100 Grams Body Weight | Total Weight of Compound in Milligrams |
|---|---|---|---|
| Lead acetate | $Pb(C_2H_3O)2.3H_2O$ | 15 | 20 |
| Lead oxide | PbO | 40 | 43 |
| Lead oxide | $Pb_3O_4$ | 45 | 49 |
| Lead tartrate | $PbC_4H_4O_6$ | 70 | 99 |
| Lead sulfide | PbS | 160 | 181 |
| Lead metal—325 mesh | Pb | above 100 | above 100 |
| Controls titanium dioxide | $TiO_2$ | Survived | 1500 |
| Controls iron oxide | $Fe_2O_3$ | Survived | 1500 |
| Controls calcium carbonate | $CaCO_3$ | Survived | 1500 |
| Controls corn oil | | Survived | 2 ml. |
| Controls distilled water | | Survived | 2 ml. |
| Controls untreated | | Survived | |

Autopsy findings were those of acute lead poisoning. In these latter animals basophilic aggregation cell counts rose rapidly and gradually fell off or were lowering at the time of sacrifice. Control animals received 1500 milligram doses of titanium dioxide, iron oxide or calcium carbonate. Additional control animals are shown in the table. Basophilic aggregation cell counts in control animals showed averages in agreement with the findings of McCord and Bradley.[23]

In Table III the lead compounds used are listed in the order of their toxicity as indicated by minimum lethal doses. When this dose is used as the toxicological index, delayed action, cumulative effects or other responses which may occur will contribute to the order of toxicity. Fairhall, Sayers and Miller[24] found that lead metal and the lesser soluble compounds assume the same toxicological importance when the ability of the animal body to store lead was used as the criteria.

By comparing Table I and Table II it is evident that both antimony metal and antimony potassium tartrate are more toxic than is lead in the form of the soluble acetate when injected intraperitoneally into albino rats at minimum lethal doses. However, under similar conditions, lead as the oxide appears to be seven times as toxic as antimony trioxide. The minimum lethal dose for lead metal was not found. Doses as large as 100 milligrams per 100 grams of body weight did not kill the animals. The body weight remained stationary with this dose over a 40-day observation period. At autopsy, unabsorbed metal was found walled off in dependent abdominal areas or caught in the omentum. Antimony metal at one-tenth this dose was fatal to rats.

### Oral Administration of Antimony

THE inauguration of a long continued feeding program, using the metal and antimony potassium tartrate, gave opportunity to observe not only the physiological response in small animals but also the distribution and retention of antimony throughout the body. The five phases of this program involved four series of albino rats and one series of rabbits, a total of 130 animals. Adequate controls were maintained for all animal groups.

The minimum lethal dose of antimony potassium tartrate by oral administration to rats was found to be 30 milligrams per 100 grams of body weight. Here again the dose given refers to the amount of antimony present in the compound. Rats survived 700 milligram doses of the metal but their weight remained stationary for many days thereafter. These materials were delivered directly to the stomach of the animals by rubber catheter and were incorporated in water and corn oil respectively.

In the second series rats were submitted to a feeding daily, except Sunday, of tablet triturates compounded in a lactose base from finely screened and thoroughly ball-milled ingredients. These animals were separated into two groups, one received treatment for six months, the other for 12 months. Tartar emetic doses were at one milligram and antimony metal doses at one or five milligrams. Feeding programs were begun on rats averaging 125 grams in weight.

In series three, rats were fed antimony ad libitum for seven and one-half months. Daily doses were one milligram for the tartrate and five milligrams for the metal.

Treatment of rats in series four was continued for 12 months. Doses of antimony potassium tartrate were gradually increased during the first six months to 10 milligrams daily per 100 grams body weight, at which level they were maintained. Similarly antimony metal was administered in gradually increasing doses up to 100 milligrams per 100 grams body weight daily. No tolerance was developed to these materials at the doses

used. This was demonstrated in a side experiment with rats where the highest doses were given without preliminary graduated step increases.

Rabbits were also given antimony ad libitum in daily doses of 40 milligrams of metal and eight milligrams of antimony potassium tartrate per kilogram of body weight. This dosage was continued four months.

**Comment**

ALL animals maintained their normal growth rates. Periodic basophilic aggregation cell counts showed normal rat averages of 2.0% and rabbit averages of 1. 8% throughout. Rats showed a slight leucocytosis. The gross and microscopic pathology of these animals simulates that previously cited for animals receving sub-lethal dosage intraperitoneally.

**Oral Administration of Lead**

THE response of rabbits given antimony differs from that elicited by lead compounds administered orally to similar animals. In a comparison study 10 rabbits each received five doses of lead compounds by stomach tube at intervals of four days. Doses were delivered in solution or suspension and the animals were submitted to autopsy from 40 to 90 days thereafter. The doses were computed in milligrams of lead present in the compound. On this basis, lead acetate, lead carbonate and lead oxide were given respectively in doses of 30, 200 and 200 milligrams per kilogram of body weight. The basophilic aggregation cell count in these leaded animals rose sharpely. Massive doses elevated this count to values as high as 15 times normal. At autopsy the gross picture was that of a typical lead poisoning.

**Chemical Determination of Antimony**

REPRESENTATIVE tissues from those animals receiving long continued oral administration of antimony were selected for chemical analyses. Various body tissues and organs including the brain, heart, lungs, liver, spleen, kidney, adrenals, bone, muscle and hair were used. The analytical method employed, using rhodamine B for the detection of small amounts of antimony, has been reported by one of us.[25] The tissue was wet ashed by treatment with hot 20% fuming sulfuric acid followed by 30% hydrogen peroxide. In 10 rabbits and 20 rats the predominant retention of antimony was in the heart, liver, kidney, lungs and bone. The hair also was found to contain slight amounts of antimony. Tissues of control animals showed the presence of antimony in amounts considerably less than in those from animals receiving antimony.

Analysis of 15 rat carcasses showed small amounts of antimony to be present. An average of one milligram was found in each carcass regardless of the daily dosage or the type of material administered. The carcasses of control animals each showed an average of one-tenth milligram of antimony as the normal constituent. Antimony apparently is not stored within the body as is lead. According to reports[2] it has been recovered in both the urine and feces following absorption.

**Spectrographic Determination of Antimony**

BODY tissues and organs from 34 representative animals receiving antimony by mouth were submitted to spectrographic determinations for antimony. The tissue was ashed and the antimony excited by a high voltage, high frequency, electrical discharge, essentially of the Tesla coil type. Pure copper electrodes were employed. The upper electrode consisted of a specially designed, inverted cone. The lower electrode was a plate. The tissue was placed on the lower plate and sparked until the sample was completely consumed. Standard samples were arranged by impregnating antimony-free tissue with known amounts of antimony compounds. Visual examination of spectrographic plates allowed a semiquantitative analysis to be made. The antimony line at 2311.5 ängstrom units was found to be satisfactory for quantitative work. A medium quartz spectrograph with a dispersion of 2 ängstrom units per millimeter in this region was employed. Eastman spectrograph analysis plates were used in this work.

Spectrographic determinations are in correlation with chemical determinations for antimony in various animal tissues and organs. High antimony retention corresponds to predominant pathological findings.

**Summary**

THE administration of common industrially used or produced forms of antimony to laboratory animals orally and intraperitoneally caused a toxic response which varied with chemical form as indicated by minimum lethal dose values. The most striking result was the high order of toxicity of the metal as compared with sulfides and of sulfides as compared with oxides.

The most important pathological effect was the consistent and invariable injury to the heart muscle, regardless of compound or dose in the concentration ranges studied. This injury was noted even at concentrations too low to produce diminution in the rate of animal growth.

Although chemical and spectrographic analysis of various body tissues of exposed animals always showed antimony in concentrations greater than those in control animals, the amount found appeared to be independent of the dose or the duration of exposure, and was never at a very high value. Apparently antimony is not stored to any extent in the organism.

Hematological findings were somewhat obscure and inconclusive. A slight eosinophilia was noted and no alteration of the basophilic aggregation cell count was produced.

Experimental comparison of the toxicology of antimony compounds with similar lead compounds showed no apparent similarity in the action of the two elements. In general antimony compounds are more toxic than similar lead compounds. Antimony, contrary to lead, appears to

be rapidly eliminated from the body without producing serious blood changes or suffering much storage.

Since all the antimony materials used in this study were of a high degree of purity, the primary effects noted could be due only to antimony. The possibility that the action might be due to lead, arsenic or other impurities present accordingly has been eliminated. In view of the definite toxic properties of antimony as shown by this and other work, its potential ability to produce occupational poisoning among exposed workers must not be disregarded. Signs of antimony intoxication in workers should be observable even when associated with lead absorption. Electrocardiographic heart studies upon antimony workers are definitely indicated.

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**Bibliography:**

1. HABECK, JOACHIM: Das Antimon, Geschichte, Gewinnung und Verwendung des Antimons in Industrie und Heilwesen sowie seine Giftwirkung. *Verof. a.d. Gebiete der Medizinalverwaltung*, 45:657, 1936.

2. CAROZZI, L.: Antimony. Occupation and Health. Brouchure 36, International Labor Office Geneva. 1:115, 1930.

3. WHITE, R. PROSSER: The Dermatergoses or Occupational Affections of the Skin. 4th ed. H. K. Lewis and Company, Ltd. London, 1934.

4. MCNALLY, WM. D.: Toxicology. *Industrial Medicine*. Chicago, 1937.

5. RAMAZZINI, BERNARDINI: De Morbis Artificum. Translated by W. C. Wright, University of Chicago Press. Chicago, 1940.

6. SCHRUMPF, P., and ZABEL, B.: Klinische und experimentelle Untersuchungen uber die antimonvergiftung der Schriftsetzer. *Arch. f. exper. Path. u. Pharmakol.*, 63:242, 1910.

7. SELISKY, A. B.: Gewerbliche Hautschadigungen durch Antimonsalze in der Textilindustrie. *Dermat. Wchnschr.*, 86:723, 1928.

8. OLIVER, SIR THOMAS: Health of Antimony Oxide Workers. *British Medical Journal*, 1381:1094, 1933.

9. FEIL, A.: Le role de L'antimoine en pathologie professionnelle; resultats d'une enguete dons une fonderie d'antimonie. *Presse med.*, 47:1133, 1939.

10. BOVERI, P. A. (Quoted by Carozzi[2]).

11. SEITZ, A.: Blood Changes in Workers in Type-Foundries. *München. med. Wchnschr.*, 70:1501, 1923.

12. SEITZ, A.: Die Hygiene im Schriftgieszereigewerbe und de experimentelle Antimonvergiftung. *Arch. f. Hyg.*, 94:284, 1924.

13. MANSON, GEO. A.: Action of Bismuth, Antimony and Others on the Circulating System. *Jour. Pharm. and Exp. Therap.*, 30:39, 1926.

14. MENEGHETTI, E.: Emopatia primitiva da solfuro di antimonio colloidale. *Haematologica*, 7:1, 1926.

15. FLURY, FERDINAND: Zur Toxikologie des Antimons. *Arch. f. exper. Path. u. Pharmakol.*, 126:87, 1927.

16. PRIBYL, EMIL: Nitrogen metabolism in experimental subacute arsenic and Antimony poisoning. *J. Biol. Chem.*, 74:755, 1927.

17. LUCIA, S. P., and BROWN, J. W.: Hematopoietic Reactions to Antimonyl Antimony. *Proc. Soc. Exper. Biol. Med.*, 31:426, 1934.

18. OELKERS, H. A.: Zur Pharmakologie des Antimons. *Arch. f. exper. Path. u. Pharmakol.*, 187, 56, 1937.

19. OELKERS, H. A., and LUDERS, H.: Antimon als Stoffwechselgift. *Klin. Wchnschr.*, 16, 680, 1937.

20. BRAHMACHARI, U. N.: Further Observations on the Toxicity of Antimonial Compounds — Delayed Antimony Poisoning. Part III. *Indian Jour. Med. Research*, 11:196, 1923.

21. CARLSON (quoted by Carozzi[2]).

22. BACHEM, A. (quoted by Carozzi[2]).

23. MCCORD, C. P., and BRADLEY, W. R.: Basophilic Aggregation in the Blood of the Newly Born. *Am. Jour. Clin. Path.*, 9:329, 1939.

24. FAIRHALL, L. T., SAYERS, R. R., and MILLER, J. W.: Relative Toxicity of Lead and Some of its Common Compounds. U. S. Pub. Health Bull. No. 253, 1940.

25. FREDRICK, WM. G.: The Estimation of Small Amounts of Antimony with Rhodamine B. (unpublished report). Presented before a meeting of the American Industrial Hygiene Association. New York, June, 1940.

# Industrial Mercury Vapor Detector

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MANY industries today utilize mercury or mercury compounds under conditions which may constitute a health hazard for the employees. The diversity of processes is shown by the fact that the manufacture of explosives, vacuum tubes, or even felt hats requires mercury, as well as do mercury boiler plants or petroleum laboratories. In all these places the mercury becomes exposed and possibly finely divided. Thus the vapor may easily be over the 0.25 mg/cu. meter toxic concentration estimated by Goodman in his excellent review of mercury poisoning.[1]

Methods made available for measuring atmospheric concentrations are two kinds — chemical or optical — and with few exceptions they are strictly for laboratory use.

Pioneer work in the development of a practical mercury detector was done in the General Electric Research Laboratory in 1926 by B. W. Nordlander.[2,3] He deposited activated selenium sulfide ($SeS_2$, a yellow powder) in a thin layer on paper. This layer darkened with the presence of mercury in the atmosphere above its surface, the reaction being a displacement of the selenium by the mercury, producing the black mercuric sulfide.

Since then, other chemical methods have been reported, but none have taken the place of $SeS_2$ in practice.

A partial list of present available chemical methods for detection of mercury in very small amounts is given.[4,11] In most of these cases the mercury is in the form of the liquid metal or one of its salts. Hence the methods are not directly applicable to the given problem.

More than 10 years ago Dr. C. W. Hewlett

developed a practical application of the optical method. This method is based upon the scattering of the "resonance" spectral line (2537A) by mercury vapor. Experiments showing this were reported by Wood about 1910.[12] This property of scattering can be demonstrated in a moderately dark room by placing a low pressure mercury vapor lamp 10 inches to 20 inches from a 6-inch x 6-inch ultraviolet fluorescent screen. The vapor from room temperature mercury, placed in a shallow dish between the two, appears as clouds of smoke on the screen.

Leighton and Leighton[13] have given an account of the optical methods, and they have shown unusual photographs of columns of vapor rising from a beaker of hot mercury, using visible and ultraviolet light.

This optical principle is applied to the detection of mercury in a transparent medium by directing the characteristic ultraviolet radiation through the medium to a phototube sensitive to 2537A radiation. The phototube yields a certain current in response to the ultraviolet radiation when the medium is free of mercury. The decrease of this response, all else being constant, means that mercury vapor is present, scattering the radiation to the walls.

A few writers have presented other or similar physical methods for the detection of mercury in the vapor state. Manley[14] gave a method for obtaining a spectrographic analysis. Hughes and Thomas[15] described a research on the absorption coefficients of mercury (from —15°C to +25°C) utilizing phototubes. This work was definitely of a laboratory type because the measuring instrument was a delicate electrometer.

Müller and Pringsheim[16] gave an application of this selective absorption. Theirs also was distinctly for laboratory use as they, too, employed an electrometer. This method was, however, for determining the mercury vapor content of air.

A recent Bulletin[17] of the N. Y. State Department of Labor has included a good summary of chemical and physical methods for estimating mercury in air.

Dr. Hewlett's detector was installed at the mercury boiler of the Hartford Electric Light Company, Hartford, Conn., where it still is in service. This installation was solely for testing flue gas and preventing the economic loss from a continued leak in the boiler. The health hazard in these mercury power plants is ordinarily low, but $SeS_2$ detectors are permanently placed at strategic locations for safety.

## Improved Design

IN developing a simpler and cheaper detector for a power plant flue gas testing, it became apparent that a portable industrial model utilizing the same basic elements could easily be made. This newly developed model was designed primarily for electrical and mechanical simplicity. However, the characteristics of maximum stability against line voltage variations, ease of dis-assembly, high speed of indication and clearing, minimum maintenance, and great flexibility of application all were considered.

The result is a detector of the optical type using a small ultraviolet lamp, and one phototube, mounted in a welded housing of 2½ inch thin-wall steel tubing. The tubing forms the "absorption chamber" which is connected only by cable to the "control panel" which in turn supports the amplifier meter and power supply. A front view appears in Fig. 1. The circuit employed is a duplex triode bridge shown schematically in Fig. 2. The meter is adjusted to zero with clear air in the absorption chamber, the balance being effected by varying resistance.

## Operation

THE introduction of a trace of mercury vapor into the chamber causes a reduction, in order, of transmitted light, photo-current, positive No. 1 grid voltage, and No. 1 plate current; and it causes an increase in No. 2 plate current and in the meter current. Thus the meter current measures the amount of light absorbed, and must reach a maximum when all the light is absorbed. This last condition can be created artificially by turning off the ultraviolet lamp. Since this maximum or "light out" meter reading is independent of lamp and phototube conditions (spacing, voltage, clean-

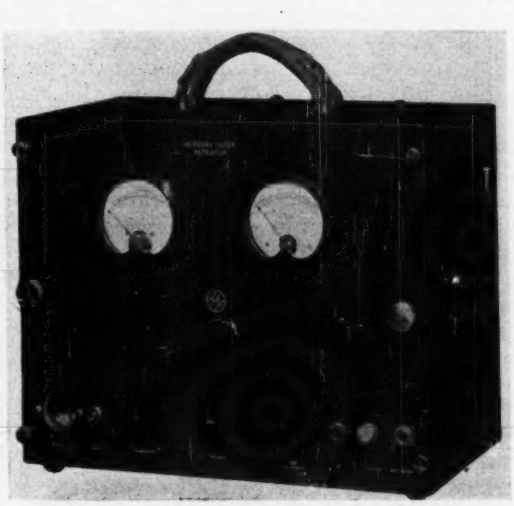

**FIG. 1.**
**General Electric Portable Mercury-Vapor Detector**

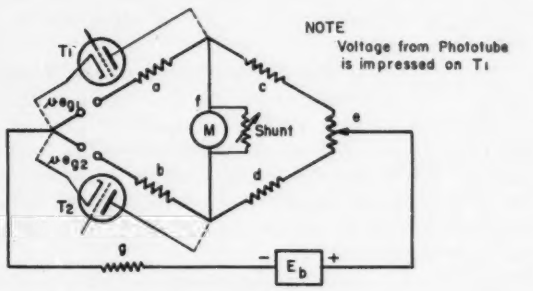


**FIG. 2.**
**Systematic Circuit**

liness, response, orientation), it is used as a basic setting. The device is calibrated by adjusting the meter to full scale on "light out" after balancing it to zero on fresh air.

**Characteristics**

FIG. 3 shows the approximate characteristics, all the family of curves being asymptotic to about full scale.

The detector under discussion, using a 10-inch chamber, has a basic calibrated range of about 0.5 to .005 part per million. Concentrations down to the one part per billion (.001 PPM) range can be measured if the sensitivity of the meter is increased by removing the meter shunt. This multiplies all readings by a factor of five. For higher concentrations the present detector can be used by diluting the specimen with fresh air in known proportions. Over 1.6 PPM cannot be measured at room temperature because the mercury will condense on the chamber walls and contaminate them.

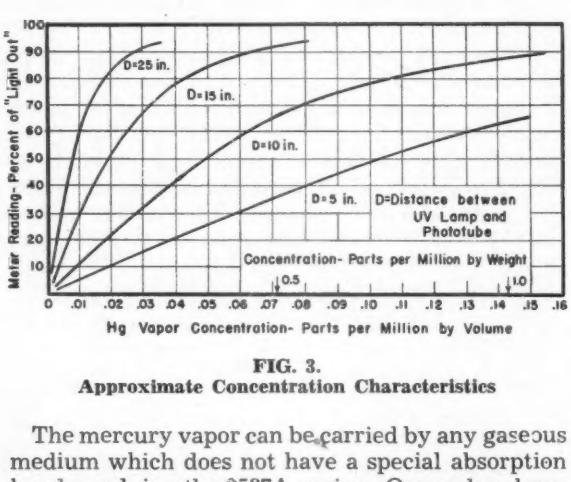


**FIG. 3.**
**Approximate Concentration Characteristics**

The mercury vapor can be carried by any gaseous medium which does not have a special absorption band overlying the 2537A region. Ozone does have such an absorption and hence affects the detector just like mercury, although to less degree. Likewise illuminating gas affects the detector, but only incomplete qualitative tests have been made with these two gases. From a group of 30 of the usual solvents, the only vapors to absorb the 2537A line significantly are benzene, pyridine, diethylacetal and toluene. Hydrogen, oxygen, ammonia, and nitrogen have been tried with negative results. Of course, smoke, fog, and dust act as physical light barriers and must be kept from the detection chamber.

The UV lamp like all low pressure arc lamps is sensitive to temperature so provision must be made to keep it within a range of ±25°F of an 80°F optimum. Also, the phototube must be kept below 150°F because the sodium coating will evaporate and cloud the window. A quartz windowed cell must be used for testing very hot or very cold gases.

The power requirement of the detector without the blower is about 60 watts. The net weight of the detector is about 25 lbs.; the panel is 11 inches x 14 inches, and the case is 7 inches deep.

**Bibliography:**

1. C. GOODMAN: *Rev. Sci. Inst.*, 9, 233 (1938).
2. B. W. NORDLANDER: *J. Ind. & Eng. Chem.*, 19, 518 (1927).
3. B. W. NORDLANDER: *Nat. Safety News, July*, 1927, p. 37. (Res. Lab. No. 418).
4. S. LOMHOLT, and J. A. CHRISTIANSEN: *Biochem. Zeits.* 81, 356 (1917).
5. A. STOCK, and R. HELLER: *Zeits. f. angew. Chemie*, 39, 466, (1926).
6. A. STOCK, and E. POHLAND: *Zeits. f. angew. Chemie*, 39, 791 (1926).
7. A. STOCK, and W. ZIMMERMAN: *Zeits. f. angew. Chemie*, 41, 547 (1928).
8. R. THILENIUS, and R. WINSTER: *Zeits. f. angew. Chemie*, 42, 284 (1929).
9. J. BODNAR, and E. SZEP: *Biochem. Zeits.*, 205, 219 (1929).
10. A. STOCK, and H. LUX: *Zeits. f. angew. Chemie*, 44, 200 (1931).
11. A. M. FRASER: *J. Indut. Hyg.*, 16, 67 (1934).
12. R. W. WOOD: Physical Optics, (3rd ed.) p. 590.
13. W. G. LEIGHTON, and P. A. LEIGHTON: *J. Chem. Educ.*, 12, 139 (1935).
14. J. J. MANLEY: *Prco. Phys. Soc.*, London, 38, Part 2 (1927).
15. A. L. HUGHES, and A. R. THOMAS: *Phys. Rev.*, 30, 466 (1927).
16. K. MULLER, and P. PRINGSHEIM: *Naturwiss.*, 364 (1930).
17. Industrial Hygiene, Div. of Ind. Hyg. N. Y. S. Dept. Labor. Vol. 18, No. 5, May, 1939, p. 235.

# Ventilation for Electroplating

*—New Data for Practical Design—*

WILLIAM P. BATTISTA, THEODORE HATCH
*and*
LEONARD GREENBURG*

CHROMIUM plating creates a serious health hazard through the exposure of operators to the chromic acid mist dispersed from the electrolytic solution. Control of this hazard is commonly accomplished by means of lateral exhaust ventilation through slots extending along the two long sides of the plating tank. Suitable manifolds and ducts connect the two slots to the exhaust fan and discharge pipe. The rate of ventilation recommended by several investigators[1,2] to reduce the concentration of chromic acid to the safe limit is determined by the relation $Q = 100\,LW$, where $Q$ = total rate of ventilation in cfm, $L$ = length of tank in feet, and $W$ = width of tank in feet.

It is clear that the rate of ventilation must vary with the tank width in such a way that a required minimum air velocity will be created at the center of the tank in all cases. According to the above equation, the relationship is taken to be a direct one; that is to say, the air velocity outward along the center line of the slot is assumed to vary inversely with the distance from the slot.

DallaValle's studies[3] of the velocity distribution around exhaust openings, however, have shown that the center line velocity in front of a suction opening is inversely proportional to an exponential value of the distance from the slot.

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*Division of Industrial Hygiene, New York State Department of Labor.

This exponent was found to have a value greater than unity. In view of this, some question may be raised with respect to the correctness of the above equation for tanks of different widths. The relationship has been investigated by the authors and the velocity contours and center line velocity equation obtained for a typical exhaust system on plating tanks. The results of this study are reported here.

In addition to the necessity for a proper relationship between rate of ventilation and tank width, it is clear that the air flow per unit of tank length should be reasonably constant throughout the entire length. This follows from the most elementary consideration of the ventilation requirements. It is not an easy matter, however, to obtain a uniform distribution of flow because space limitations often do not permit the use of the elaborate exhaust piping which is required. In practice, there is need for a simple and compact ventilating system which, with reasonable power cost, will provide a constant rate of ventilation for each foot of tank length. A second objective of the present study therefore was to develop the design requirements for such a system.

Two plating tanks of different widths may be said to have equally effective ventilation when the velocity contours over the centers of the two tanks have the same values. Hence, the rate of ventilation must increase with tank width in accordance with the relationship which governs the change in velocity with distance outward from the slot.

**Ventilation Rate and Tank Width**

THEORETICALLY, the velocity distribution may be considered from the standpoint of two limiting conditions: (1) a slot of infinite length or, in effect, a line source of suction of constant value throughout its length; and (2) a suction opening of such limited dimensions that the conditions approach those of a point source of suction. In the first case wherein an unobstructed line of equal suction throughout its length is assumed, the velocity contour surfaces will take the form of a series of concentric cylinders with the line source as an axis. Under this condition, the air velocity in the space around the line source will vary directly with the distance away from the line since the surface area of cylinders of equal length varies directly with their radii. Applying this to the ventilation of a plating tank, the following relation could be written:

$$Q = KLW \text{ or } Q/L = KW$$

*where* $Q$ = air volume per unit time;
$K$ = a constant;
$L$ = tank length;
$W$ = tank width.

This equation has the same form as the one now in use.

In the second limiting case wherein approximately a point source of suction in an unobstructed space is assumed, the velocity contour surfaces will take the form of a series of concentric spheres. The surface areas of these contours will vary with the square of their radii, thus:

$$A = KR^2$$

*where* $A$ = surface area of spherical contour
$R$ = radius of contour spheres

Considering $R$ in terms of tank width, the relation between rate of ventilation and tank width becomes

$$Q/L = KW^2$$

The velocity distribution in the actual case of an exhaust slot on a plating tank does not conform to either of these limiting cases but lies somewhere between the two. It follows, therefore, that the exponent of $W$ in the relation $Q/L = KW^a$ must have a value between 1 and 2. For tanks of a relatively great length, $a$ will approach unity whereas for short tanks the exponent will have a value closer to 2.

DallaValle[3] has developed the following general equation for the relation between the center line velocity $V$ at various distances $X$ from the exhaust opening and the velocity, $V_0$ through the opening:

$$\frac{V}{V_o - V} = KX^{-a}$$

A determination of the exact value of the exponent $a$ for a typical exhaust slot was made by the authors on apparatus described later in this report (Arrangement *E* in Fig. 3).

Velocity measurements were taken by means of the thermal anemometer in a vertical plane normal to the midpoint of a horizontal slot 6 ft. long by 1 in. wide having an approximately uniform rate of air flow throughout its length. A smooth flat surface placed horizontally 7 in. below the slot simulated the electrolytic surface in a plating tank.

Velocity contour curves according to accepted procedure[3] corresponding to the common slot velocity of 2000 fpm for chromium plating are shown in Fig. 1. (These contours are for one slot only. For two slots on opposite sides of the tank, the combined contours would be equivalent to the vectorial sum of the contours shown in Fig. 1 and a reverse set of the same relative values established by the second slot.) Center line velocity values, outward from the slot, were obtained from these curves and plotted according to DallaValle's general equation in Fig. 2. The slope of the straight line resulting from this plot indicates a value of the exponent $a$ of 1.625 for the 6 ft. slot. For other slot lengths the exponent will have different values between the limits of 1 and 2. In practice, however, plating tanks do not vary greatly in length and the exponential value of 1.6 may therefore be employed in the equation $Q/L = KW^{1.6}$ to determine the rate of ventilation in relation to tank width for most plating tank problems (NOTE—The velocity distribution characteristics of exhaust slots have been subjected to more elaborate dimensional analysis by Silverman,[4] the results of which will provide valuable information relative to the ventilation requirements

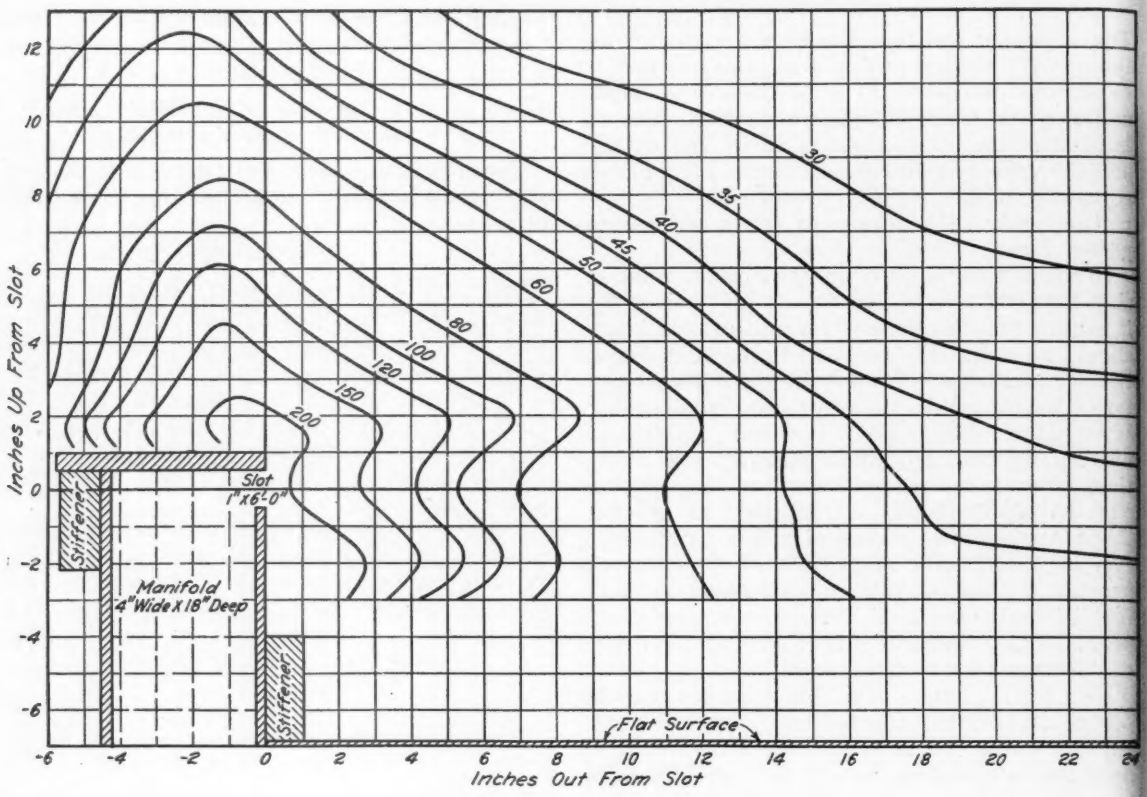


**FIG. 1.**
**Velocity contours for plating tank exhaust slot**

for the many different process tanks encountered in industry.)

The limited data on ventilation requirements for chromium plating tanks of different widths[1,2] are not sufficient to show the correctness of the new or earlier equations. It is to be noted, however, that the rate of ventilation and the power consumption for wide tanks are greater when determined by the proposed relationship than by the equation now in use. Thus, if a rate of ventilation of 100 cfm per sq. ft. of tank area is necessary for a tank 3 ft. wide, the exponential formula would require a rate of 160 cfm per sq. ft. for a tank 4 ft. in width. In the interests of economy in design, therefore, further study of the ventilation requirements on plating tanks is needed, particularly with reference to the value of the constant *K*.

### Design of Exhaust Manifold

THE need for a uniform rate of ventilation through the entire length of the exhaust slot has been pointed out. This is most nearly secured by using a slotted plenum chamber of relatively great cross-sectional area in which the negative pressure is substantially uniform regardless of the location of the fan connection. Another method is to provide numerous pipe connections from the fan to equally spaced points along a slotted duct. Neither scheme is practical because of the limited space generally available around plating tanks. A tapered duct with a slot of uniform width with the fan connected to the larger end is found in practice to give a non-uniform distribution of flow. The use of an adjustable tapered slot is objectionable because the slot opening is seldom properly adjusted or fixed permanently in the correct position.

The problem becomes one of designing a compact manifold of simple construction which will provide a reasonably uniform distribution of flow and does not occupy too much space. The simplest design is one which employs a manifold of uni-

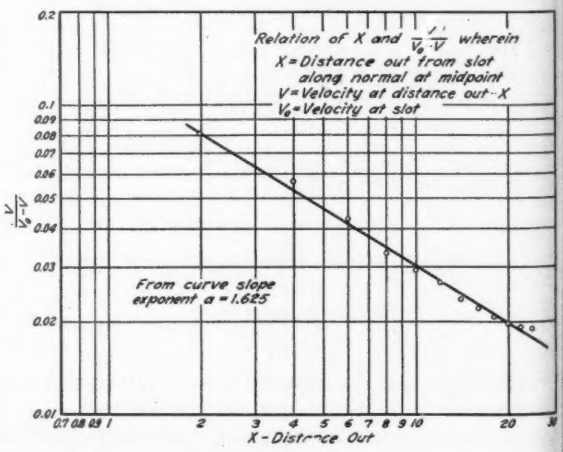


**FIG. 2.**
**Relation between center line velocity and distance outward from slot**

form cross-section on the two long sides of the tank with a common exhaust chamber and fan connection at one end. The cross-sectional dimensions of the manifold should be as large as possible in order to approach the effect of a plenum chamber. In accordance with common practice in ventilation, the manifold may be provided with internal vanes, as required, to insure a uniform distribution of flow. The design requirements of such a manifold are developed below.

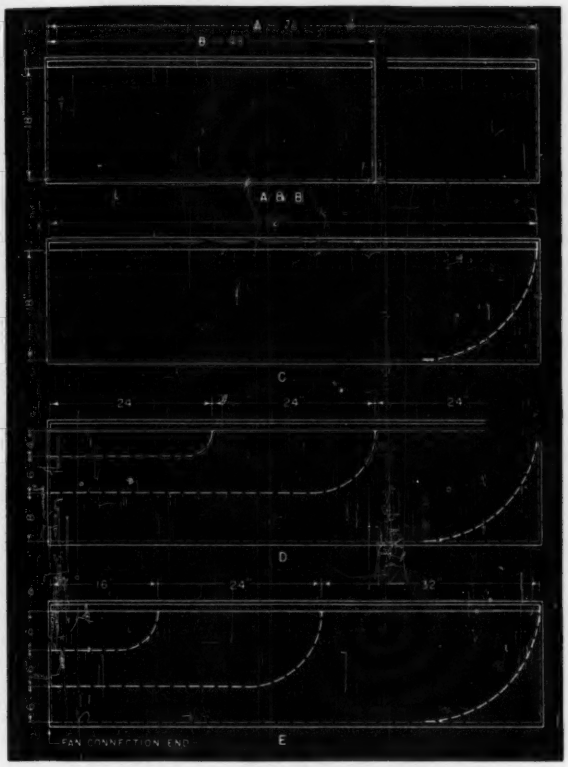


**FIG. 3.**
**Side views of test manifolds**

The test apparatus was fabricated of one-quarter inch plywood and one-half inch lumber with stiffening members, as required. Internal dimensions of the manifold were 72 in. long, 18 in. deep and 4 in. wide. A one-inch slot ran the full length of the manifold at the top of one side, thus providing an air intake area of 0.5 sq. ft. A plywood box 30 in. long, 27 in. high and 12 in. wide was joined at right angles to the manifold to serve as an exhaust plenum chamber. The fan was connected to this plenum at the rear top corner diagonally remote from the manifold with common 8 in. pipe. The additional height of plenum over that of the manifold served to simulate the air flow effect of the tapered elbow ordinarily used in practice to connect the fan pipe to the two manifolds.

A blast gate on the exhaust fan was adjusted throughout the tests so that the average air velocity through the slot was 2000 fpm.

Velocities at the slot were measured with a direct reading air velocity meter using the appropriate attachment. Readings were made at the top, middle and bottom of the slot for each one-inch interval along the slot. These were averaged to obtain one set of readings for the one-inch slot opening.

The first series of velocity readings were obtained with the simple manifold (Fig. 3*A*). The results

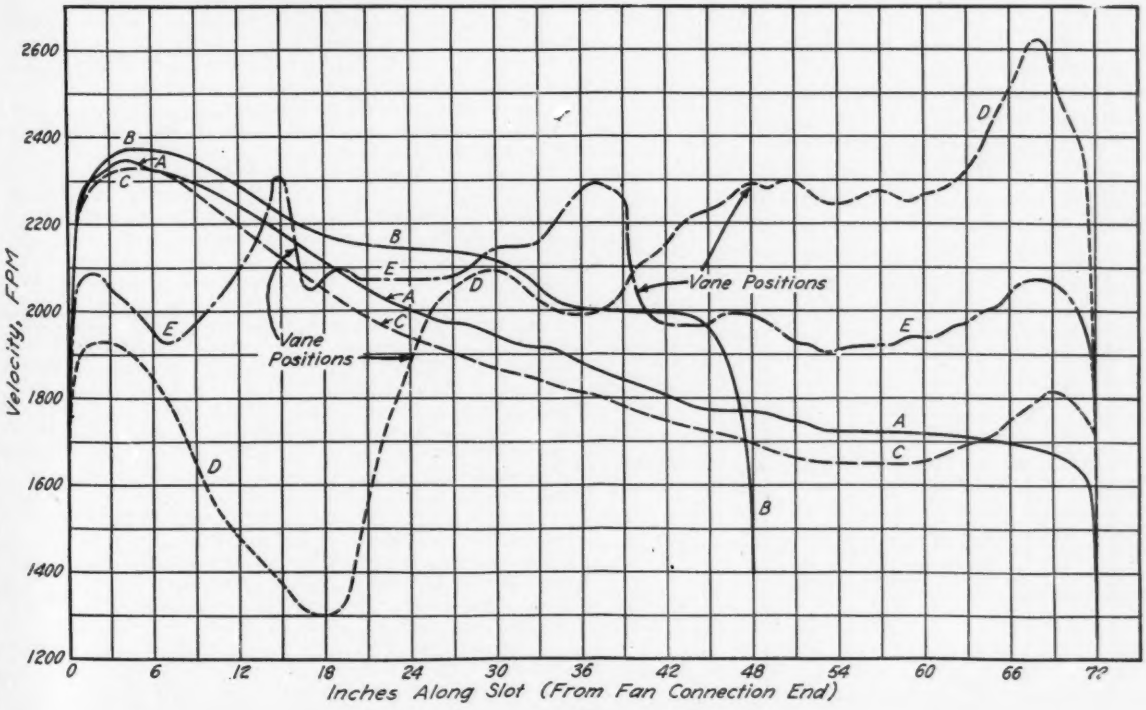


**FIG. 4.**
**Velocity distribution along slot for the various test manifolds**

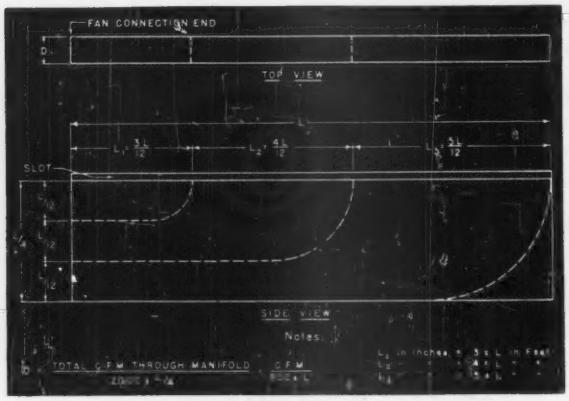


**FIG. 5.**
**Recommended design of exhaust manifolds for uniform distribution of flow**

are plotted as Curve *A* in Fig. 4 and reveal a marked decrease in slot velocity with distance from the exhaust end of the duct. In the second test, the manifold was shortened to 4 ft., thus making its cross-sectional dimensions relatively greater and more nearly approaching the plenum chamber effect (Fig. 3*B*). The velocity distribution, plotted as Curve *B* in Fig. 4, also shows a marked non-uniformity along the slot. The results of these two tests are in agreement with past experience and indicate that a simple uniform manifold of the limited dimensions permitted in practice is not acceptable. A single curved vane at the remote end of the manifold (Fig. 3*C*) served to increase the flow in this region, as shown in Curve *C* in Fig. 4, but did not significantly improve the distribution of flow through the slot as a whole. The results of this test indicate clearly the need for additional distributing vanes.

Accordingly, two more curved partitions were installed in such a way as to divide the manifold into three separate sections, as shown in Fig. 3*D* and 3*E*. In Arrangement *D*, the vanes were equally spaced along the slot and arbitrarily located vertically so as to provide the greatest cross-sectional area in the bottom section which draws air from the remote end. The resulting velocity distribution, shown by Curve *D* in Fig. 4, indicates that too much air was drawn through the bottom section. Consequently, the vanes were relocated, as in Fig. 3*E*, to provide equal vertical spacing and a varying distribution along the slot according to simple fractional proportions. With the exception of minor disturbances in flow in close proximity to the vanes, this arrangement provides a reasonably uniform slot velocity (Curve *E*, Fig. 4). Undoubtedly, further investigation would show more desirable vane arrangements, but the practical features — and particularly the simplicity — of arrangement *E* recommends it. The final design, with the proportions adjusted to require only the simplest design calculations, is shown in Fig. 5. The arrangement shown in Fig. 5 may be employed for manifolds of any reasonable length, provided all dimensions are determined in the proportions shown. For long tanks this requires considerable depth to the manifold. A more compact arrangement for long tanks is given by installing fan connections at both ends of the tank thus making the effective manifold length for each fan one-half the tank length.

**References:**

1. BLOOMFIELD, J. J., and BLUM, WM.: Health Hazards in Chromium Plating, Public Health Reports, 43, 2330, 1928.

2. RILEY, E. C., and GOLDMAN, F. H.: Control of Chromic Acid Mists from Plating Tanks, Public Health Reports, 52, 172, 1937.

3. DALLAVALLE, J. M.: Principles of Exhaust Hood Design, U. S. Public Health Service, Washington, 1939.

4. SILVERMAN, LESLIE: Harvard School of Public Health, personal communication.

# News and Notes

## —*of Association Activities*—

THE writer finds that he lacks information concerning some of the sectional activities, and has been unable to fill in this information, owing to a change in the publication policy of this Journal.

However, the ever-active New York group held a very successful meeting January 15, 1941, at the District Health Center Building in New York, with DR. ALEXANDER O. GETTLER, Professor of Toxicology and Chemistry, New York University, as the principal speaker. DR. GETTLER's topic was "Micro-analytical Methods in Chemistry," and he explained the use of these methods in connection with the New York City work in which several thousand bodies are examined in cases of suspicious and sudden death. In the course of his talk, he described micro methods for the distilling of volatile liquids from brain matter and the determination of carbon monoxide, mercury and fluorides. He also outlined the micro methods used in his laboratory for the fractional distillation of volatile solvents and determinations of molecular weights and boiling points of liquids. At the next meeting, in the Director's Room of the McGraw-Hill Building, March 5, 1941, DR. LYDIA G. GIBERSON spoke on the subject of "Psychiatric Aspects of Industrial Hygiene."

The Chicago Section had MR. B. F. POSTMAN, Industrial Hygiene Engineer in the Connecticut State Department of Health, as a speaker on January 30, 1941. The subject "Engineering Measures for the Control of Industrial Health Hazards" was completely illustrated with lantern slides and provided a very interesting outline of various actual installations of preventive measures.

The Chicago Section heard the writer, on March 27, 1941, discuss "Control Measures as Found in Large Industry."

The Michigan Section combined with the symposium which was held at the University of Michigan in Ann Arbor January 23, 1941, to have an evening meeting. This plan, which we believe merits consideration by other sections, involves having one or two meetings a year in some other city than the one in which the majority of the members reside. DR. C. P. MCCORD gave a very interesting and frank discussion of "The Role of Industrial Hygiene in National Defense."

The next meeting of the Michigan Section, held Thursday, March 20, 1941, was different in that it was not a dinner meeting. However, the topic was about the same, with the subject matter entirely different. DR. CLARENCE SELBY discussed "The Role of Industrial Hygiene in National Defense" as he sees it from his position as Chairman of the Sub-committee acting as an advisory body on health matters pertaining to industry.

The writer would be interested in securing the views of members concerning their cooperation in sending in items of industrial hygiene importance for inclusion in this column.. —GORDON C. HARROLD, *Secretary*.